Bandolier 57

What do we think?
What do we know?
What can we prove?

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*The views expressed in **Bandolier** are those of the authors, and are not necessarily those of the NHSE Anglia & Oxford*

BEST AVAILABLE TRUTH

Whenever "evidence" is discussed, arguments often break out which seem to ***Bandolier*** to be much like baying for the moon. If a review chooses only randomised trials, then what about other types of evidence? If a randomised trial is chosen, then is the trial big enough to draw implementable conclusions? Are case-control or cohort studies good enough, other than from which to develop hypotheses for future testing? What about ethics? What should we do next?

It all seems impossible if every sentence ends with a question mark and every conclusion is "more research". We live in a real world and have to make the best decisions we can. We were helped by a thoughtful Irish biochemist who said that what we are seeking is the "best available truth".

That may be from a great systematic review - as in this issue on analgesics in dysmenorhoea. It may be from a single great randomised trial with useful outcomes - as in this issue on second cataract surgery. It may be from epidemiological work - as in this issue looking at the links between homocysteine and heart disease. But if we know our source is the best available truth, and we update that truth regularly, then we can be confident that we're likely to be doing OK.

That is why electronic libraries are so important, because they can be relatively a painless way of updating ourselves. The Cochrane Library is one, but there are other ways, and electronic library / information resources are delivering the goods, and it is difficult to remember how we lived without them.

Change of address

Those who want to notify a change of address can help us by giving the address to which Bandolier is sent now as well as the new address - it really helps us. If you can, please send the address label on the envelope as well, as Bandolier may be mailed from one of several sources.

ANALGESICS FOR DYSMENORRHOEA

Dysmenorrhoea affects many women of reproductive age, and is a frequent cause of time lost from work or school as well as interfering with daily living. Treatment is usually with NSAIDs and minor analgesics. A systematic review [1] tells us how effective these are for primary dysmenorrhoea.

Search

Randomised trials were sought using a number of search strategies, including requests for information from manufacturers. Studies had to be randomised, and primary dysmenorrhoea was defined as a history of painful menstrual cycles and exclusion of organic causes by physical examination.

The main outcome was pain relief of at least moderate intensity, and the main comparison was with placebo. Secondary outcomes were women needing rescue analgesics, women experiencing restriction of daily living and women experiencing absence from work or school. Adverse effects were also examined.

Results

Most trials were double-blind of either parallel or cross-over design, predominantly comparing the test analgesic with placebo.

Randomised trials of pain relief for dysmenorrhoea

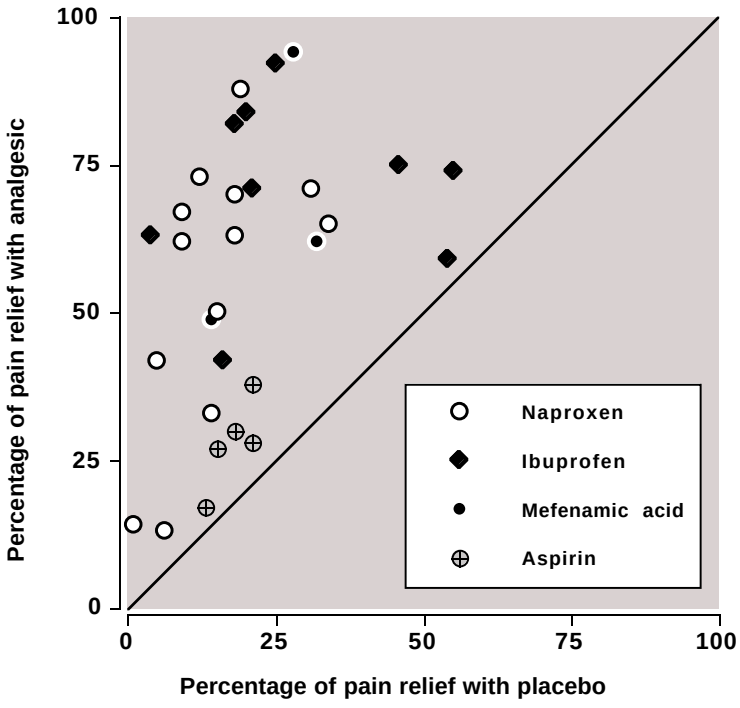


Table 1: Effectiveness of analgesics for pain relief of primary dysmenorrhoea

Analgesic	Number of Trials	Number of patients	Percent improved with analgesic	Percent improved with placebo	NNT (95% CI)
Naproxen	13	1706	59	17	2.4 (2.2 to 2.7)
Ibuprofen	9	599	70	31	2.6 (2.2 to 3.2)
Mefenamic acid	3	518	64	31	3.0 (2.4 to 4.0)
Aspirin	5	416	29	18	9.2 (5.3 to 35)

Pain relief

The results for pain relief are shown in the Figure and Table 1. Compared with placebo, naproxen (550 or 275 mg four times daily), ibuprofen (400 mg four times daily) and mefenamic acid (250-500 mg four times daily) had numbers needed to treat of between 2.4 and 3.0, with overlapping 95% confidence intervals, indicating no real difference between them. Five trials of aspirin (650 mg four times daily) had a much higher NNT of 9.2, with no overlap of confidence intervals with the NSAIDs. One comparison between paracetamol (650 mg four times daily) and placebo showed no difference between them.

Restriction of daily living

Women taking naproxen or ibuprofen were less likely to have restrictions of daily living (Table 2). The NNTs were 3.8 (3.2 to 4.6) for naproxen and 2.4 (1.9 to 3.2) for ibuprofen. Aspirin did not have this beneficial effect, and the point estimate for the NNT was 8. There were no data on mefenamic acid.

Absence from work or school

Naproxen reduced greatly (by about 70%) the amount of time away from work or school (Table 2). The NNT was 3.9 (3.3 to 4.6). One study on ibuprofen mirrored this effect, and one study on aspirin did not have this beneficial effect. There were no data on mefenamic acid.

Adverse effects

Adverse effects were mainly nausea, dizziness and headache. There was a suggestion that naproxen caused more adverse effects (mainly nausea), but the power of studies to detect this was low and confidence intervals wide.

Comment

This is a well-done systematic review which demonstrated that naproxen, ibuprofen and mefenamic acid are effective. Aspirin was less effective and paracetamol 650 mg was not effective in a single study. The authors conclude that, based on efficacy and absence of common adverse effects, ibuprofen is probably the treatment of choice.

The only complaint one could have is the issue of dose. It would be helpful to know the NNTs at particular dose levels, though we suspect that the authors reflect what they found in the original reports.

Bandolier *wishes to thank the authors for making original data available.*

Reference:

1 WY Zhang, A Li Wan Po. Efficacy of minor analgesics in primary dysmenorrhoea: a systematic review. British Journal of Obstetrics and Gynaecology 1998 105: 780-9.

Table 2: Effectiveness of analgesics on reducing restriction of daily life and absence from work or school caused by primary dysmenorrhoea

Analgesic	Number of Trials	Number of patients	Percent affected with analgesic	Percent affected with placebo	NNT (95% CI)
Reducing restriction of daily living					
Naproxen	7	1341	60	86	3.8 (3.2 to 4.6)
Ibuprofen	3	234	12	55	2.4 (1.9 to 3.2)
Aspirin	3	203	50	62	8.0 (3.8 to >100)
Reducing absence from work or school					
Naproxen	7	1345	8	34	3.9 (3.3 to 4.6)

HOMOCYSTEINE AND HEART DISEASE

There is a growing recognition that high levels of homocysteine are associated with heart disease. This started in the late 1960s when a pathologist in Boston encountered two children with homocystinuria, who, despite being very young, had advanced atherosclerosis, though the plaques contained no lipid. The pathologist concerned, Kilmer McCully, was given a hard time for putting forward the suggestion of a possible link between homocysteine and the formation of atheromatous plaque [1].

Basis of homocysteine theory

Homocysteine is a curious sulphur-containing amino acid formed during methionine metabolism. It can dimerise to homocystine, or form disulphide bonds with proteins to form so-called "protein-bound" homocysteine. In plasma about 80% of homocysteine is protein bound.

Metabolism of homocysteine is by pathways which re-methylate it (and which require vitamin B12 and folic acid), or by a trans-sulphuration pathway which requires vitamin B6. Homocysteine in blood (and elsewhere) is a product of how much methionine is eaten, mainly in protein (with about three times more methionine in animal than plant protein), and how much is metabolised (and metabolism may be affected by amounts of B vitamins and folate available).

What homocysteine does to arteries

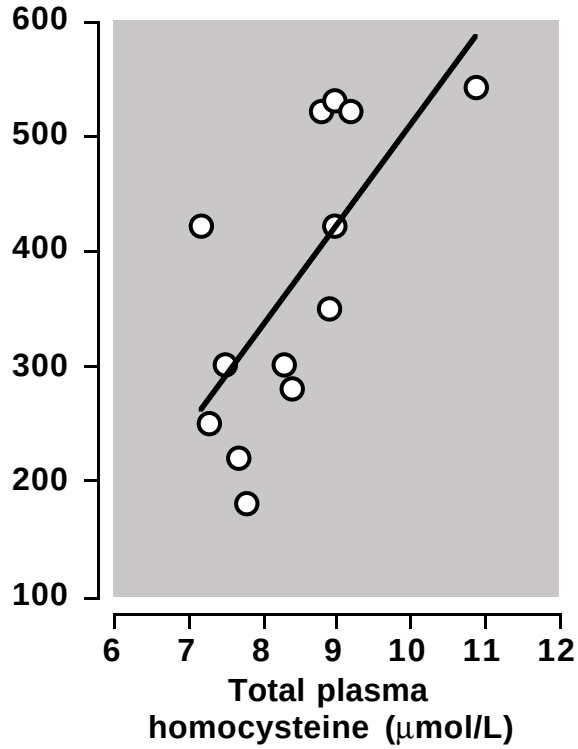
Too much homocysteine and the result is atherosclerosis. Evidence for this comes from a variety of sources:

- Homocysteine generates superoxide and hydrogen peroxide which have been linked to damage to arterial endothelium.
- Homocysteine changes coagulation factor levels so as to encourage clot formation.
- Homocysteine prevents small arteries from dilating so they are more vulnerable to obstruction.
- Homocysteine causes smooth muscle cells in the arterial wall to multiply.
- Homocysteine, when infused into animal arteries, caused the linings to slough off and produce lesions much like human atheromas.
- Homocysteine has a reactive product, homocysteine thiolactone, which interacts with low density lipoproteins causing them to precipitate and damage endothelial tissue.
- Homocysteine thiolactone causes platelets to aggregate.

Homocysteine, the epidemiology

There is a growing amount of evidence associating high homocysteine levels ('normal values' are usually regarded as being below about 14 μmol/L) with death from heart disease. A classic study (Figure 1) showed that high homocysteine levels in a population were associated with higher levels of cardiovascular disease mortality [2].

Figure 1: Association between cardiovascular disease mortality and plasma total homocysteine [2]



In the BUPA prospective study of 21,500 men aged 35 to 64 years, homocysteine in stored samples was compared in 229 men without a history of ischaemic heart disease and who subsequently died of ischaemic heart disease over a mean follow-up of 8.7 years, and 1126 age-matched controls [3]. This study showed that men in the highest quartile of plasma homocysteine levels (over 15 μmol/L) were nearly three times more likely to die of ischaemic heart disease, even after adjusting for factors like apolipoprotein levels and blood pressure (odds ratio 2.9, 95% confidence interval 2.0 to 4.1).

The BUPA study also contained data from other prospective and retrospective studies which examined homocysteine levels and fatal or nonfatal cardiac events. There was considerable consistency, with an odds ratio of ischaemic heart disease for each 5 μmol/L increase in serum homocysteine of 1.8 (1.5 to 2.2) in a total of about 2,300 subjects.

Another prospective examination of the relationship between plasma homocysteine and mortality was carried out in 587 patients with angiographically confirmed coronary artery

Mortality over four years in 587 patients with coronary artery disease [4]

Homocysteine (μmol/L)	Mortality (%)
<9	3.8
9-14.9	8.6
≥15	24.7

disease [4]. Over a median follow up of 4.6 years 64 patients died. For those patients whose plasma homocysteine was <9 μmol/L, only 3.8% died, compared with 25% of those with plasma homocysteine ≥15 μmol/L (Table).

Homocysteine and vitamins

Serum homocysteine concentrations have been correlated with plasma vitamin concentration and vitamin intake in 1160 adult survivors, aged 67 to 96 years, from the Framingham study [5]. Homocysteine was higher in men and women aged over 80 years, but after adjusting for age, sex and levels of other vitamins, there was a strong inverse correlation between plasma homocysteine and folate. Individuals with the lowest plasma folate were twelve times more likely to have an elevated homocysteine concentration (defined as being above 14 μmol/L) after adjusting for age, sex and other B vitamins.

The mean serum homocysteine concentration and the proportion of individuals with elevated plasma homocysteine (>14 μmol/L) was correlated with B vitamin intake (Figure 2). The quintile with the lowest B vitamin intake had a prevalence of homocysteinaemia of 53%. Similar results were found for a vitamin index expressing serum concentrations of B vitamins.

That there is a relationship between increased intake of vitamins, especially B vitamins, and heart disease was confirmed in the Nurses' Health Study [6]. In 14 years of follow up from 1980 there were 658 nonfatal and 281 fatal cases of coronary heart disease in over 80,000 women who gave detailed information on diet and vitamin supplements at entry. After controlling for cardiovascular risk factors, the incidence of heart disease in those with the highest intake of folate was 31% lower than those with the lowest intake. For vitamin B6 those with the highest intake had a 33% lower risk of disease. For women in the highest quintile for both folate and vitamin B6, the risk of heart disease was reduced by 45% (95% confidence interval 26% to 59%).

Therapeutic implications

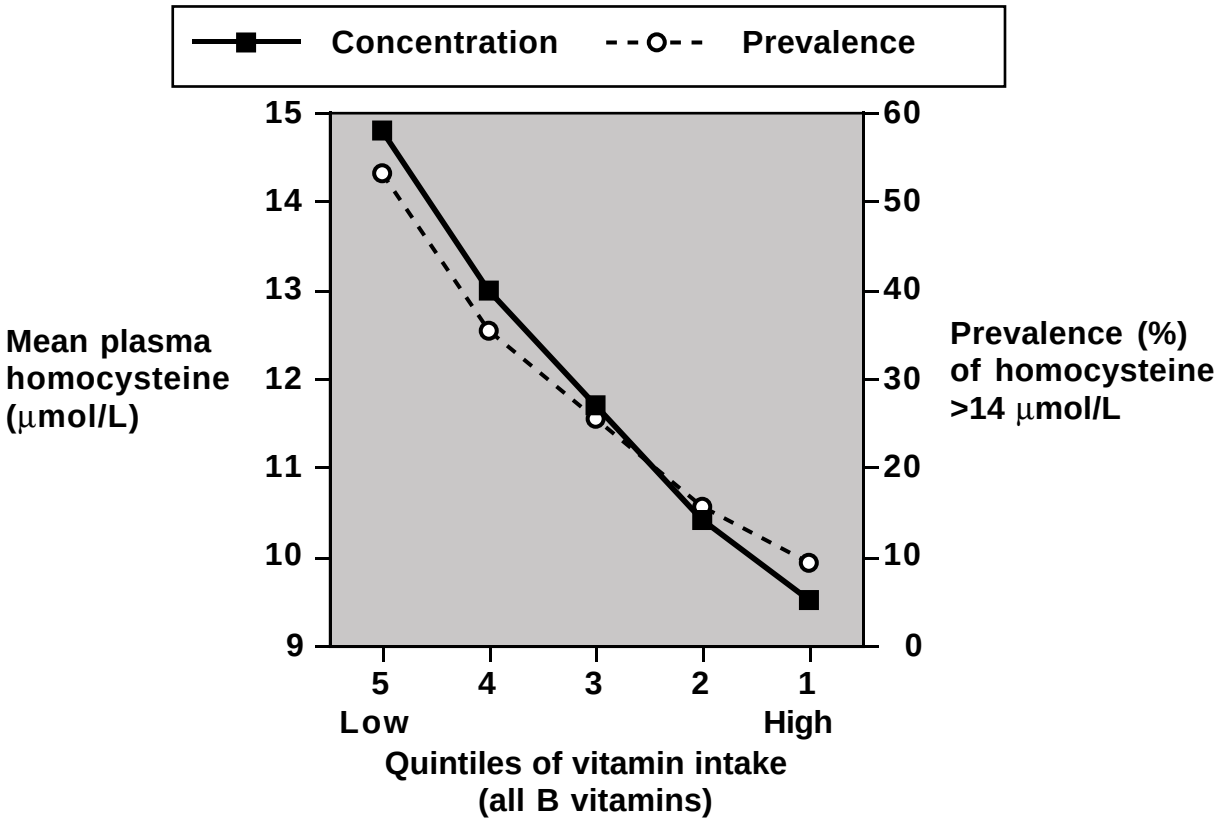
There are few randomised controlled trials of interventions to reduce homocysteine concentrations in blood and correlation of that with clinical outcome. All we have is an observation [7] that treatment with folate and B vitamins stops the increase in atherosclerotic plaque area in patients with unexplained atherosclerosis which might have a genetic cause and who have homocysteine concentrations of >14 μmol/L.

Comment

Homocysteine is clearly important, and the epidemiological evidence linking high levels of homocysteine in blood to increased rates of heart disease is becoming impressive. A homocysteine industry is starting to develop, in which new and easier methods for analysing this amino acid are being developed and marketed. Right now assays aren't so easy, but before long we will probably be seeing people comparing their homocysteine level just as they now discuss their cholesterol or PSA. Another screening test to worry about for someone!

The nutritional advice seems to be not much different from anything else we have seen before. Eat lots of fruit and vegetables, don't be too liberal with animal protein, avoid refined carbohydrates and take alcohol in moderation. There is definitely a school of thought, though, that folate and B

Figure 2: Elevated homocysteine concentrations by B vitamin intake



vitamin intakes should be supplemented - "eat right and take a multivitamin" seems to be the emerging consensus.

References:

1 Kilmer McCully: pioneer of the homocysteine theory. Lancet 1998 352: 1364.
2 G Alfthan, A Aro, KF Gey. Plasma homocysteine and cardiovascular disease mortality. Lancet 1997 349: 397.
3 NJ Wald, HC Watt, MR Law et al. Homocysteine and ischaemic heart disease. Archives of Internal Medicine 1998 158: 862-7.
4 O Nygård, JE Nordrehaug, H Refsum et al. Plasma homocysteine and mortality in patients with coronary artery disease. New England Journal of Medicine 1997 337: 230-6.
5 J Selhub, PF Jacques, PW Wilson et al. Vitamin status and intake as primary determinants of homocysteinaemia in an elderly population. JAMA 1993 270: 2693-8.
6 EB Rimm, WC Willett, FB Hu et al. Folate and vitamin B6 from diet and supplements in relation to risk of coronary heart disease among women. JAMA 1998 279: 359-64.
7 JC Peterson, JD Spence. Vitamins and progression of atherosclerosis in hyper-homocyst(e)inaemia. Lancet 1998 351: 263.

SECOND CATARACT SURGERY

Surgery to remove cataracts has moved on apace, so that now many of these operations are done as day cases. Cataract surgery works, and is a major benefit to most people, generally elderly, who need it. Cataracts are usually bilateral, and there has apparently been some argument that if surgery on one eye is successful, then surgery on the other is not needed. A randomised trial of second cataract surgery from Bristol refutes that [1].

Study

The usual waiting time for second cataract surgery in Bristol was seven to 12 months. The study randomised patients needing second cataract surgery (without other ocular comorbidity) to either expedited surgery for 98 patients (within six weeks) or routine waiting time for 94 patients. Before randomisation and about six months after surgery detailed assessments were carried out. These included four primary outcomes of binocular visual function, and four daily living outcomes asking about:

- difficulties with reading,
- eyesight preventing individuals from doing things they wanted,
- an overall description of vision,
- whether eyesight problems interfered with life.

Results

Patients in the two groups were similar at randomisation. All eight primary outcomes were highly significantly better in the group with expedited surgery - with statistical significance generally at the 1 in 10,000 level. The proportion of patients having poor binocular vision (stereoacuity 3000 or worse) was dramatically lower at 12% in those who had expedited surgery compared with 70% in those still waiting for surgery.

The numbers needed to treat for the four outcomes describing daily living are shown in the Table. Second eye cataract surgery prevented eyesight interfering with life quite a lot or a great deal in one of every four patients having the operation.

Comment

The daily living outcomes represented responses which were at the harsh end of possible answers, and appear to be sensible representations of real life. The questions were arrived at through an intensive process to make sure that they made sense and that patients could make sense of them. This was a well done trial which answers the question: second cataract eye surgery benefits patients.

Reference:
1 DA Laidlaw, RA Harrad, CD Hopper et al. Randomised trial of effectiveness of second eye cataract surgery. Lancet 1998 352: 925-9.

Primary outcomes of daily living in trial of second cataract surgery

Outcome	Percent with surgery	Percent without surgery	NNT (95% CI)
At least some difficulty reading normal print	6	36	3.5 (2.5 to 5.5)
Eyesight preventing activities most or all the time	0	11	9.4 (5.8 to 24)
Below average overall vision	0	18	5.5 (3.8 to 9.9)
Eyesight interfering with life quite a lot or a great deal	1	26	4.1 (3.0 to 6.5)

BENZODIAZEPINES AND CRASHES

The proportion of over-65s in the population will increase. After adjusting for the number of miles driven, crash rates in the elderly population are twice as high as for middle-aged drivers, and only under-25s have higher crash rates. Over-65s often take benzodiazepines for insomnia or anxiety. Benzodiazepines affect the central nervous system in a number of ways, and the elderly may be less able to metabolise the drugs, so one consequence of all this could be reduced ability to drive safely. Two studies have now shown an association between benzodiazepine use and increased risk of motor vehicle crashes.

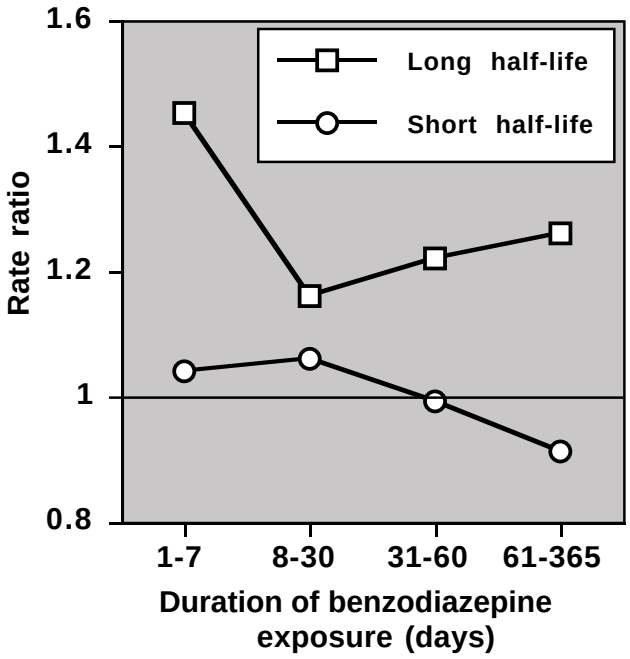
Elderly drivers in Quebec

The first [1] was conducted in Quebec and examined 225,000 people aged 67 to 84 years who were drivers of cars in the years 1990 to 1993. There were 5579 drivers involved in a crash in which someone was injured, and a random sample of controls, from which 55790 control person days were selected. Benzodiazepine exposure was ascertained from records of prescriptions filled, a method which had previously been validated. Cases and controls were similar.

Short half-life benzodiazepines were not associated with any increased risk of motor vehicle crashes, with an overall rate ratio of 0.96 (95% confidence interval 0.88 to 1.05). There was no increased risk in the first week of exposure, nor at any longer period (Figure 1).

Long half-life benzodiazepines, in contrast, were associated with a 28% increased risk of a crash - rate ratio 1.28 (1.12 to 1.45). The increased risk was associated with all durations of benzodiazepine use (Figure 1), but this was highest and statistically significant for the first week (45% increased risk) and use longer than two months.

Figure 1: Rate ratio for motor vehicle crashes by duration of exposure and benzodiazepine half-life



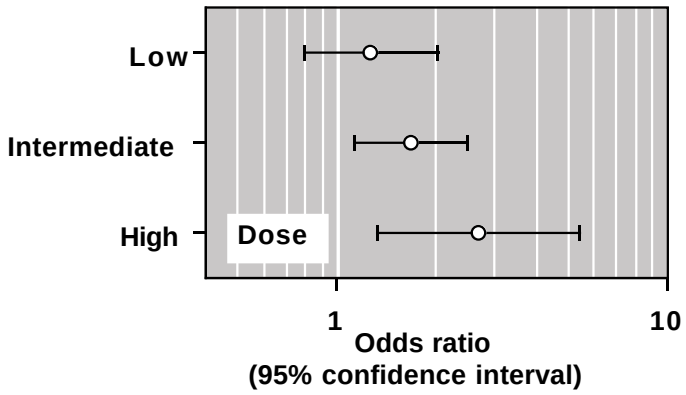
In this report long half-life (> 24 hours) benzodiazepines included clonazepam, diazepam, clorazepate, chlordiazepoxide, flurazepam and nitrazepam. Those with a short half-life (≤24 hours) were alprazolam, bromazepam, lorazepam, oxazepam, temazepam and triazolam.

Benzodiazepines in Scotland

A study in Tayside looked at prescribed drugs in drivers aged 18 years and over who had a first road traffic accident attended by Tayside police between mid-1992 and mid-1995. There were 19,386 cases. Using a record linking database, cases acted as their own controls. The odds of having a crash while exposed were compared with those while not exposed.

The results showed that of the drug classes looked at (benzodiazepines, tricyclic antidepressants, SSRIs and other psychoactive drugs), only benzodiazepines were associated with increased risk of a crash. There were 235 individuals exposed to benzodiazepines who had a crash, with an odds ratio of 1.62 (95% confidence interval 1.24 to 2.12). There was a clear dose response (Figure 2), with high-dose benzodiazepines increasing risk of a crash by 170%.

Figure 2: Odds ratio for road traffic accident by dose of benzodiazepine



Comment

These two thorough studies underline that benzodiazepines can be associated with higher risks of road accidents. Higher doses and long acting benzodiazepines will be the principal culprits. Benzodiazepine use has apparently been associated with increased fall and hip fracture rates in the elderly, especially with longer acting compounds. Reducing benzodiazepine prescribing in general practice may be one strategy, and in ***Bandolier*** 4 we highlighted a simple way to reduce the number of prescriptions in general practices with established policies to minimise benzodiazepine prescribing. By using a letter and information sheets a fifth of patients stopped taking benzodiazepines altogether and 40% reduced their prescriptions by half.

References:

1 B Hemmelgarn, S Suissa, A Huang, JF Boivin, G Pinard. Benzodiazepine use and the risk of motor vehicle crash in the elderly. JAMA 1997 278: 27-31.

2 F Barbone, AD McMahon, PG Davey et al. Association of road-traffic accidents with benzodiazepine use. Lancet 1998 352: 1331-6.

The Joint Department of Primary Care & Population Sciences at UCL and Royal Free Medical Schools are pleased to announce the Fourth London Workshop on Teaching Evidence-Based Health Care

1st - 5th February 1999 -Organiser: Dr Trish Greenhalgh

Based on the model developed at McMaster University Canada, the multi-disciplinary residential workshop offers delegates the chance to learn, and learn how to teach others, the skills of evidence-based health care in tutor facilitated small groups. The programme includes access to hands-on database search clinics and step-by-step tuition on statistical procedures.

For further details and an application form please contact Marcia Rigby on any of the following:

e-mail:	ebp@ucl.ac.uk
website:	http://www.ucl.ac.uk/primcare-popsci/uebpp/ttt.htm
post:	Department of Primary Care, Archway Site, Whittington Hospital, London N19 5NF
fax:	+44 (0)171 281 8004

CASP

Multi-media route to learning critical appraisal skills

Many healthcare professionals and those in related industries struggle to read and understand research articles. CASP has undertaken 350 workshops attended by over 6000 people, teaching healthcare professionals and lay members of committees how to search, find, critically appraise research papers and then put into practice evidence-based healthcare decisions.

The contents of the workshops have been transformed to make this process easier using a multi-media CD-ROM and an open learning resource. They encompass the activities and knowledge required to make effective healthcare decisions based on "good" research-based evidence and put them into practice. The advantages of open learning resources include:

- √ they can be accessed at a time and place suitable for the individual
- √ they will act as a reinforcement of learning by having many different and practical cases, tasks and activities to work through
- √ they will act as a pre-workshop introduction to those with a much lower baseline knowledge
- √ they are accessible to a much wider audience
- √ they are important tools for higher education establishments.

The cost of the CD-ROM is £30 before November 30th and £35 thereafter. For the open learning resource the prices are £55 before, and £65 after November 30th. Contact (Fax) CASP on +44 1865 226959 or Update Software (Fax) on +44 1865 516918.

STRESS URINARY INCONTINENCE IN WOMEN

Two systematic reviews published in recent years provide some basis on which to consider how to deal with this difficult and common problem. The overall prevalence of urinary incontinence in women is about 30%, but probably less than 5% in men. Stress urinary incontinence is the involuntary loss of urine during physical exertion, like coughing, sneezing, laughing, running, or lifting.

Surgical treatment

A systematic review of surgical interventions [1] found 11 randomised trials, 20 non-randomised studies or prospective cohort studies and 45 retrospective cohort studies. It found that the methodological quality of the 31 prospective studies was weak, and that the variability in inclusion criteria, surgical management and assessments of outcomes meant that no combining of data was possible.

The conclusion, therefore, was that evidence for the effectiveness of surgery was weak and conclusions speculative. It stressed particularly the lack of reliable information on complications after surgery and safety.

Conservative treatment

A systematic review of conservative treatments [2] found 24 randomised trials, of which 11 were of sufficient quality to allow for some analysis (though with lots of different methods and few studies for any one treatment). The main finding was that there was good evidence of benefit from pelvic floor exercises compared with no treatment, but that the amount of evidence to justify other modalities, like higher levels of exercise, or use of electrostimulation or intravaginal resistance devices, was low.

Comment

These reviews both comment on the generally poor quality of studies and lack of good quality evidence, but they are a good start for anyone trying to produce guidelines. Pelvic floor exercises which strengthen pelvic floor muscles are thought to improve the sphincteric action around the urethra and support the pelvic organs. It is suggested that strong contraction of the pelvic floor muscles will 'clamp' pressure rises in the urethra and prevent stress urinary incontinence. Pelvic floor exercises seem to work. The one message that comes through loud and clear is the need for good quality trials in which information on inclusion (diagnostic) criteria, therapy, and outcomes which include benefit and harm are clear and unambiguous.

References:

1 NA Black, SH Downs. The effectiveness of surgery for stress incontinence in women: a systematic review. British Journal of Urology 1996 78: 497-510.

2 LC Berghmans, HJ Hendriks, K Bø et al. Conservative treatment of stress urinary incontinence in women: a systematic review of randomized clinical trials. British Journal of Urology 1998 82: 181-91.

Book reviews

Dictionary of Evidence-based Medicine. A Li Wan Po. Radcliffe Medical Press, Abingdon, 1998. 165pp, £16.50. ISBN 1 85775 305 4.

The first, and perhaps appropriate, item in this dictionary is "Ability to pay", something ***Bandolier*** always worries about. But if you want to know what a Likert scale or Jarman index is, then you'll find a brief description plus a useful reference in this book.

Which of us hasn't from time to time (or even more frequently than that) been stumped by some weird piece of jargon? Can you define an ASTRO-PU. Well, with this book on your shelf, it is a simple matter to figure out that it is relevant and simple, and that if you need to go to the font of all knowledge on the subject, then it's in the BMJ.

Bandolier is in there, nicely placed between "balanced design" and "Bayes, Thomas R". It is interesting to know that his fame derives from a posthumously published paper. There are many things you'll know, of course, but even more that you won't. Hands up all those who know what Berkson's fallacy is? (A spurious correlation which may be observed between two diseases or between a disease and a risk factor arising from biased sampling.)

There will be a few definitions in this dictionary with which the sophisticated would wish to quibble, but not many. The style is simple and direct, and for those of us with few or tired neurones, it's on the button.

A good stocking filler this, and the price, at about ten pence a page, is well within most people's ability to pay.

Evidence-based Healthcare. A Practical Guide for Therapists. Tracy Bury & Judy Mead. Butterworth Heinemann, Oxford, 1998. 249 pp, £19.99. ISBN 0 7506 3783 8.

This is a really good book directed towards physiotherapists. It is clearly written and it covers all the bases. There are some chapters on change management that are particularly interesting (and the case studies useful), and excellent stuff on how to handle the findings and using of evidence.

Any physiotherapist new to the ideas of EBM, or anyone else for that matter, will find this book useful and stimulating. Those who work in the area already will find little new, but might find the way some of the ideas are conveyed rather well done.

One quibble, though. The book lacks examples. There are some examples of evidence, but nowhere near enough. Now that possibly reflects a dearth of evidence in the physical therapy area, but there are others from pharmacology and some forms of complementary medicine which could be used. A harder edge through examples would do more to challenge ingrained habit or lack of resolve. Perhaps time is of the essence here, and with more therapists engaging in research and implementation of evidence, the second edition will have more meat.

But that is a quibble, and the reality is that the care and attention lavished on this book is obvious. It is an important contribution in an important area.

The Science of Presenting Well. Ian Wilkinson. AACC Press, Washington 1998. 109pp, US$15. ISBN 0 915274 94 9.

"Information has two key characteristics: its content and its means of transmission. The basic units of information content are ideas, or 'memes'. Memes are the information world's equivalent of genes. Like genes, memes are bits of information passed from individual to individual. And like genes, they love to reproduce."

The transmission of "memes", though, is the key to this amusing, informative and useful book. Ian Wilkinson seems to have been there, done that, and got the T-shirt. He's a Hitchhiker fan, and his introduction is datelined 'Phobos'.

If you have read Bill Bryson, and found yourself laughing aloud on a plane or train, then you will have already experienced the slight embarrassment you can expect if you follow Ian Wilkinson's thoughts in public. But this book isn't all about laughs: it uses laughs to get ideas over, and to help prepare both the naive and the experienced presenter. Perhaps most of all it helps anyone who is going to give a talk, or who has given many, to laugh at themselves. Pomposity is punctured!

The chapter on oral presentations is wonderful. He tells us that fear of public speaking is top of the list of phobias, with death down at number seven. He tells us how to get over the problem, how to structure a talk, how to work the audience, and what to avoid. Like the laser pointer! Once you see this referred to as the 'Jedi-Knight Syndrome', you will never wave it around so mindlessly again. That's another plus from the book - using it as a scoring system when you are bored to death with yet another poor presenter.

This is a classic. If you thought nothing ever good came out of clinical biochemistry, then on this, at least, you would be wrong. It's great.

If you can't get it in bookshops, try the AACC Press Internet site at www.aacc.org.

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